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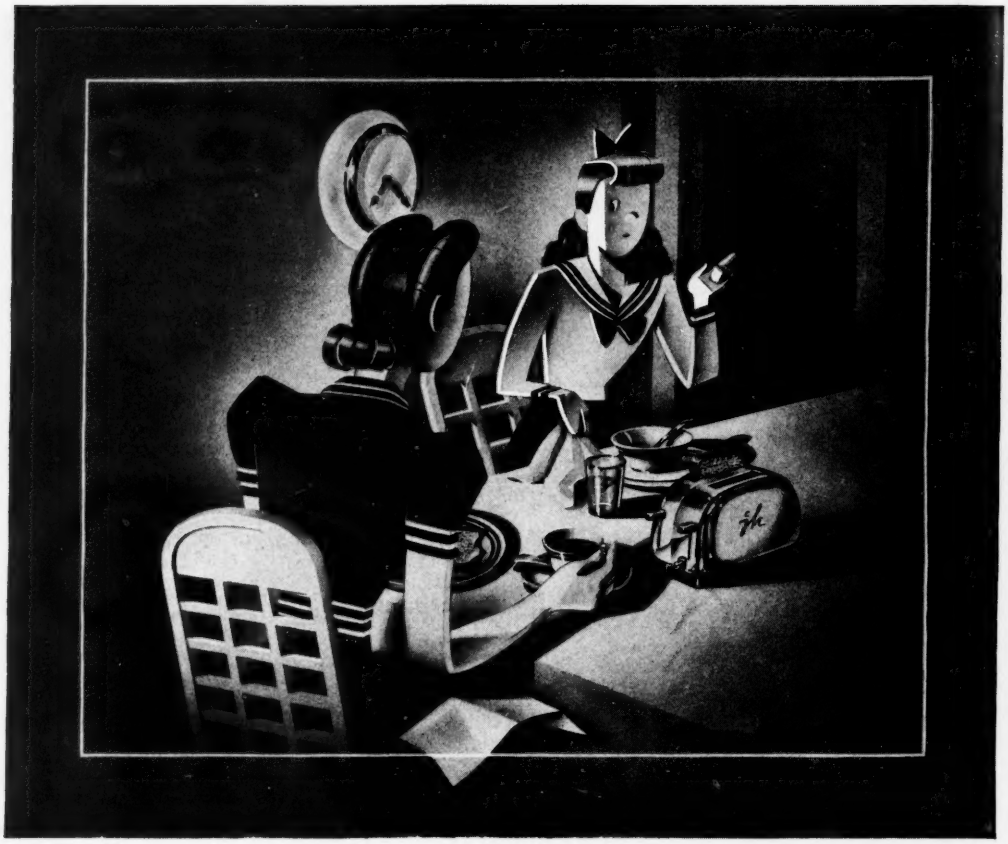

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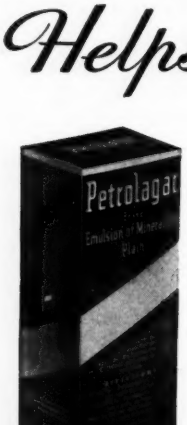

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THE RHODE ISLAND MEDICAL JOURNAL

Volume XXIV MAY, 1941 Number 5

VARICOSE VEINS AND RELATED CONDITIONS

I. THE MODERN TREATMENT OF VARICOSE VEINS

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The three basic methods of relieving varicose veins, namely (1) sclerosis, (2) compression, and (3) ligation have been known for a long period. Obliteration in situ has been traced to Hippocrates[1] ("the varix itself is to be punctured in many places") while ligation was performed by Celsus as early as the first century. Excision or stripping advocated a number of years ago were almost completely replaced by simple injection and more recently by ligation and injection. Proper treatment requires an understanding of the anatomy and pathologic physiology.

Anatomy and Physiology.

There are three groups of veins in the lower extremity, (1) the superficial, (2) the deep and (3) the communicating veins. The superficial group lies outside the deep fascia. The deep veins accompany arteries or ramify among the groups of muscles. The superficial veins are connected with the deep by a set of communicating veins which perforate the muscular aponeurosis.

The superficial and deep veins are furnished with valves so arranged that blood will flow past them only in one direction, namely toward the heart. The communicating veins of the calves have valves so disposed that blood can flow through them only from the superficial system toward the deep. The venous circulation in a dependent extremity is motivated mainly by muscular activity which propels the blood past these valves.

A varicosity is defined as an abnormally dilated superficial vein characterized by loss of function of its valves. The superficial veins of the leg belong generally to two great systems, (1) the long or internal saphenous and (2) the short or external saphenous. The long saphenous vein (Fig. I), after its origin on the medial aspect of the foot, passes up the medial aspect of the leg and thigh to the fossa ovalis, through which it enters the femoral vein at the sapheno-femoral junction. Just before emptying into the femoral vein it is joined by several important tributaries (1. the external pudendal, 2. the superficial circumflex iliac, and 3. the superficial epigastric and sometimes others). The importance of these tributaries will be discussed with the technique of high ligation.

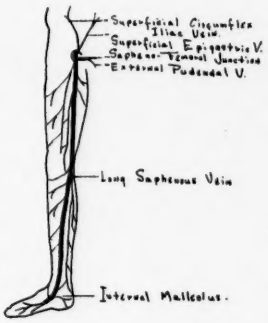

Fig. I. The Long Saphenous System

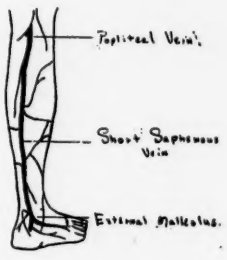

Fig. II. The Short Saphenous System

The short saphenous vein (Fig. II), after its origin on the lateral aspect of the foot, gradually assumes a more median position and ascends the posterior surface of the leg, almost in the mid-line until it perforates the popliteal fascia to communicate with the deep system. Varicosities of the short saphenous vein are comparatively unusual.

An important etiological factor seems to be an hereditary predisposition. Careful family histories will reveal an appreciable familial incidence. A number of other factors, of course, may contribute. The superficial veins have very little support in contrast to the deep veins which are enclosed by muscle and fascia. In the female pregnancy is commonly observed to be the precipitating cause. Re-

From the Peripheral Vascular Clinic, Beth Israel Hospital, Boston, Mass. Read before the Providence Medical Association, Jan. 6, 1941.

cent work on venous pressures[2] has emphasized the importance of the erect posture as a predisposing factor. Furthermore, varicose veins result from preceding thrombophlebitis which may damage or destroy valves.

Clinical.

Before treatment is begun every patient should be studied carefully to eliminate other possible pathologic states, e.g., syphilis, diabetes and peripheral arterial disease. It is well to remember, too, that peripheral edema even when accompanied by large varicose veins may be of cardiac or nutritional origin. Ulcers may be trophic or neurogenic in origin, the result of spinal cord disease or spina bifida. Recently chronic ulcers of the leg associated with congenital hemolytic jaundice have been described;[3] these ulcers are usually located near the medial malleolus (a location typical of varicose ulcers) and are permanently cured only when the underlying blood dyscrasia is relieved by splenectomy.

After these preliminary studies the exact anatomic involvement is investigated in order to determine the course of treatment. The patient is examined in the standing position. Visual inspection is of some value in estimating the extent of involvement and frequently will disclose the location of the incompetence. Palpation of the extremity, however, is much more accurate, and will frequently disclose varices previously unsuspected.

Attention should then be directed to the following:

1. Whether the valvular incompetency occurs in the long saphenous, the short saphenous or the communicating veins, and
2. Whether the deep veins are patent.

Several tests have been devised which facilitate this study.

The Trendelenburg test[4] and its modifications provide the answer to the first question. To grasp the significance of these tests it is convenient to visualize the communications between the systems as shown in Fig. III. The long saphenous veins and the deep veins communicate proximally at the sapheno-femoral junction and distally by way of the communicating veins. The short saphenous vein communicates with the deep veins at the popliteal space. The arrows show the normal direction of blood flow.

The patient is allowed to lie down and the leg is then elevated until the veins have been emptied. A tourniquet is placed high on the thigh. The patient is then asked to stand with the tourniquet held in place. If the veins do not fill within a half minute the deep and short saphenous systems may be disregarded as contributing factors. The tourniquet is then released and the vein is seen or felt to fill rapidly from above downward. This demonstrates that the major source of leakage is proximal to the tourniquet and that the valves of the long saphenous system are incompetent.

If, however, there is filling before the tourniquet is removed the source of leakage must be distal. In this case, either the short saphenous system or the communicating veins have incompetent valves.

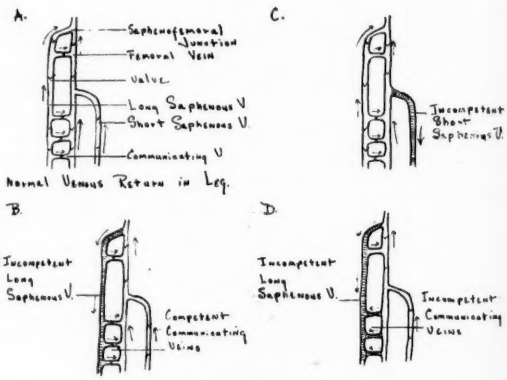

Fig. III.

To distinguish between these two latter groups the tourniquet is reapplied just below the knee. If now the veins do not fill with the patient standing, they are of short saphenous origin. If filling occurs there are incompetent communicating veins in the lower leg. It is desirable in some cases to determine the level of incompetent communicating veins more precisely by performing the tourniquet test at various levels.

Although most observers agree that a permanent occlusion of the deep veins following phlebitis is quite unusual it is well if in doubt to establish their patency before obliterating the superficial ones. The superficial veins are emptied by elevation, and then an elastic bandage is applied from toes to knee. If the patient while walking about experiences no discomfort or cramps with the veins compressed the deep veins may be considered as patent.

On the basis of these tests Faxon[5] has classified all cases in six groups, as follows:

1. Cases in which there is a demonstrable incompetent main internal saphenous trunk above the knee.
2. Cases in which there is incompetence of the valves of the deep perforating branches without ulceration or brawny edema of the lower leg.
3. Cases in which there is incompetence in the valves of the deep perforating branches with ulceration or brawny edema of the lower leg.
4. Cases in which there is a demonstrable short saphenous trunk originating in the popliteal space.
5. Cases in which the varicosities have followed a deep phlebitis and are shown to be compensatory in nature.
6. Cases in which dilated veins and flares are found unassociated with incompetence of the valves in either the main trunk or the perforating branches.

This classification forms a rational basis for treatment.

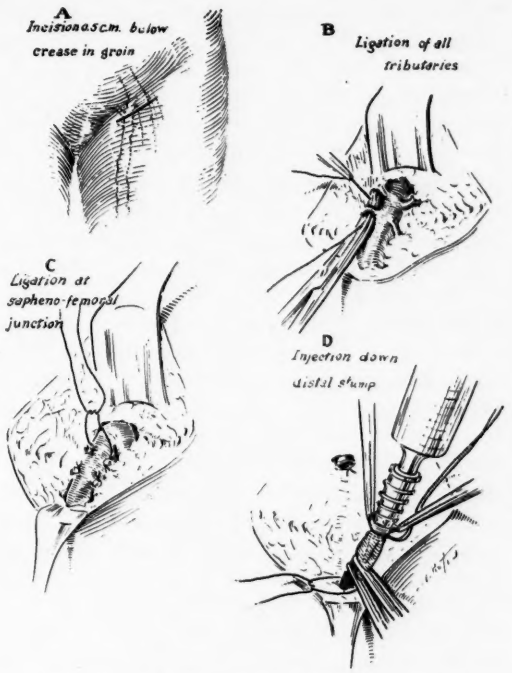

Fig. IV. Technique of High Saphenous Vein Ligation and Retrograde Injection

GROUP I. (*Incompetent Long Saphenous Vein with Competent Communicating Veins.*) In 1933 Faxon[6] reviewed 314 such cases treated by injection alone. 63% of the total number showed recurrence in an average period of 1.4 years, and 25% showed new varicosities. In a total of 102 cases similarly treated Martenssen[7] found 55 relapses within a maximum period of one and a half years.

In contrast to these results Faxon and Barrow[8] reported follow-up data on 365 high saphenous vein ligations in 228 patients. They found satisfactory or perfect results after at least one and a half years in 80% of the operations. Sears and Cohen[9] report almost identical results from the Beth Israel Hospital, Boston, in 135 operations after a follow-up of 2 to 6 years. In both groups causes of failure were usually obvious and should be largely eliminated in the future by a more meticulous evaluation of individual patients.

The results of these studies point to high ligation of the long saphenous vein as the procedure of choice of this group.

Technique of High Ligation with Retrograde Injection. (Fig. IV.) Under local anaesthesia a 5 cm. incision (longer if necessary) is made 0.5 cm. below the crease of the groin centered just medially to the pulsation of the femoral artery. The fat and fascia are divided so as to expose the trunk of the long saphenous vein as it terminates at the sapheno-femoral junction. Incisions at a lower level, as often recommended, render exposure of the sapheno-femoral junction appreciably more difficult. All of the tributaries entering the long saphenous vein are cut and ligated. It is important to recognize all of these because they are a potent source of recurrence. There are usually at least three in number, as previously noted. The main trunk is then divided close to the femoral vein and the distal segment is injected retrogradely with 10 to 30 c.c. of Varisol. This is a proprietary mixture of invert sugar and sodium chloride which has been used successfully by both of the groups mentioned. A Faxon cannula as illustrated may be conveniently used for this purpose. Pratt[10] has recently recommended inserting a small catheter for the retrograde injection to insure a more uniform distribution of the sclerosing agent. Following this 1 cm. of the vein is excised and both stumps are ligated. Post-operative injections with sodium morrhurate or quinine and urethane are usually necessary.

GROUP II. (*Incompetent Communicating Veins without Ulceration or Brawny Edema.*) Here likewise a high saphenous vein ligation is indicated. Treatment of the communicating veins may be postponed. But the patients should be followed carefully, and in the event of the appearance of trophic changes or of recurrent varicosities, the communicating veins should then be ligated.

GROUP III. (*Incompetence of the Communicating Veins Accompanied by Brawny Edema or Ulceration.*) For adequate treatment in this group ligation of the communicating veins as well as high saphenous vein ligation are necessary. Existing ulcerations must be healed before dealing with the communicating veins. The treatment of ulcers will be considered separately. Linton[11] has been able, on the basis of anatomical studies, to divide the communicating veins into three clinical groups: the medial, the anterior and the lateral. The medial group, the most commonly involved, is affected in 80 to 90 per cent of cases. The others are involved in 5 and 15 per cent of cases respectively.

Technique of Ligation of the Communicating Veins. When the medial group of veins is involved a broad curved flap bearing the skin, fat and fascia is reflected. The communicating veins are thus cut across and tied, and the flap is resutured in place. The skin flap must be handled with meticulous care to prevent necrosis. When the fascia shows considerable scarring it is desirable to excise the involved portion of the fascia before closing the skin.

When several groups of communicating veins require treatment it is preferable, as suggested by Linton, to reflect the skin and fascia through straight incisions. These are placed so as to correspond to the involved group and extend from the level of the malleoli to the upper third of the leg.

GROUP IV. (*Incompetent Short Saphenous Trunk.*) Marked dilatations of the short saphenous system are best treated by ligation and injection.

GROUP V. (*Varicosities Following Deep Phlebitis which are Compensatory in Nature.*) In the rare event that persistent occlusion of the deep veins is demonstrated by the tests no treatment of the varicosities is indicated.

GROUP VI. (*Dilated Veins and Flares without Incompetence of the Main Trunks or the Communicating Veins.*) Varicosities of limited extent or isolated dilated veins may be treated by injection alone. In the case of flares a feeder vein may sometimes be injected. Generally, however, treatment of flares is unsatisfactory. They are, however, rarely the cause of complications and are usually only of cosmetic interest.

Technique of Injection. The method whether or not accompanied by operation is the same. Many modifications have been described in the literature, but there is not much evidence that the exact method of injection matters greatly. At the Beth Israel and Massachusetts General Hospitals the injection is performed usually with the patient standing and without tourniquets or occluders. About 2 c.c. of sodium morrhuate or quinine and urethane are injected with a fine hypodermic needle, following which a pressure dressing is applied. The injections are begun at a distal point and progress proximally. They are made at intervals of one week.

Summary.

1. Proper selection of treatment for varicose veins depends upon a careful evaluation of the individual case.

2. Tests have been described to determine the competency of the long saphenous, short saphenous and communicating veins, and patency of the deep veins.

3. In properly selected cases ligation plus injection has proved superior to injection alone.

4. The techniques have been described for high saphenous vein ligation, injection, and ligation of the communicating veins.

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II. ULCERS, PHLEBITIS, AND THE THROMBOSES.

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For the purpose of this discussion, ulcers will be divided into two general groups; (1) Varicose and (2) Post-phlebitic.

Varicose Ulcers.

These ulcers are definitely related to varicose veins and are usually found in the region of the inner ankle, frequently overriding the lower end of a column of regurgitant blood. Trauma initiates most of these ulcers; the alleged role of fungi in etiology has not been clearly established.

The most important factor for *cure* is the obliteration of the offending varicosities by compression, injection, or ligation and injection. But *healing* of the ulcer is the patient's first concern.

For economic reasons, in many of our patients, it is necessary to attempt healing by ambulatory methods. We rely on some form of compression. When the ulcer base is foul and dirty, the patient is taught to apply warm wet compresses (salt or boric solution) under a moisture-retaining wrapping of oil silk, all covered by a woven bandage from toes to knee.

When the ulcer becomes cleaner, a simpler routine is used. Some patients are taught to do a daily dressing as follows: Wash the ulcer with soap or peroxide solution, apply a bland ointment on gauze, cover the latter with a large rubber sponge, then apply the woven bandage from toes to knee.

More popular than the above are the two proprietary types of bandage which compress the foot and leg, including the ulcer, and are left in place for one to three weeks. The first consists of a woven bandage coated on one side with adhesive, (Elastoplast or Ace adhesive). This is particularly useful when there is a tendency to edema, but should not be used when there is a dermatitis or sensitivity to adhesive. The second type is a gauze bandage impregnated with a modified Unna's zinc oxide-gelatin mixture (Gelocast), thus encasing the lower extremity in a bland rubbery cast. Our preference is for the latter method, even when there is extensive dermatitis, because it is non-irritating.

Heaped-up granulations, when present despite compression, are gently cut or curretted from the healing border. Silver nitrate is avoided because it destroys epithelial cells and thus delays healing.

Patients with large or badly infected ulcers are advised to go to bed, for the surest and quickest way to heal any ulcer is by bed rest, elevation and warm moist compresses.

If the patient has been hospitalized, the saphenous vein is ligated and divided on the day before discharge. Skin grafting is advantageous when the ulcer is large.

Occasionally we have encountered a painful ulcer with an undermined edge from which mixed aerobes and anaerobes have been cultured. The use of a 40% zinc peroxide suspension has resulted in rapid healing and cessation of pain.

Rarely it has been necessary to crush the saphenous nerve because of a very painful ulcer in the region of the internal malleolus. The nerve is exposed either in Hunter's canal or just below the knee.

Other therapeutic adjuncts, occasionally used, are cod liver oil ointment, obtundia cream and gentian violet solution (2% aqueous).

Post-Phlebitic Ulcers.

These occur as a late sequel of ilio-femoral thrombophlebitis ("Milk leg"). Usually there is residual edema of the lower extremity plus induration and pigmentation of the antero-medial portion of the lower leg. The ulceration appears from six months to ten years after the original "Milk leg" and is usually preceded by a non-suppurative local inflammation. There is involvement of skin, fat and fascia; the recurrent attacks of cellulitis leave much cicatrization which interferes with local nourishment and thus with healing.

These indurations and ulcers frequently lie over incompetent communicating veins and may be accompanied by dilated superficial veins. The latter are usually compensatory in nature, appearing subsequent to the original deep thrombosis.

Case History: M. M. is a thirty-two year old woman who suffered a "Milk leg" eight years ago, after a normal delivery. Her extensive white swelling gradually lessened but never completely disappeared. Four years ago a small ulcer appeared

just above the internal malleolus (Fig. 1) and healed rapidly when she was hospitalized. This area repeatedly breaks down into a chronic painful ulcer when she is ambulatory. There are no palpable nor visible varicosities.

Figure 1

Case History: R. S. is a thirty-six year old man. An appendectomy ten years ago was complicated by a "Milk leg" for which he was kept in bed seven weeks. Since then, his leg has always been moderately swollen. Six months ago a painful indurated area above the internal malleolus broke down and now he has a sluggish ulcer almost identical with that in figure 1. Two other findings are of interest. There is an extensive fungus infection of his groins and he has large varicosities on his leg, thigh, and abdominal wall. These are, of course, compensatory veins.

All ulcers require great patience and should be treated conservatively. When the futility of such treatment is finally realized in a given case, surgery is indicated. Block excision of the indurated area, down to and including the fascia, plus skin grafting is radical but quite certain. (Fig. 2.) More recently we have used a modified "flap" operation (Fig. 3) which permits the division of communicating veins and the excision of most of the related fascia. The removal of this fascia, much of which is thickened as a result of earlier cellulitis, permits the improvement of the blood supply of the remaining flap of skin and fat. Through the upper end of the incision the saphenous vein may be divided, if indicated, and the saphenous nerve may be crushed.

Phlebitis.

Four types will be discussed briefly.

1. *Femoro-iliac thrombophlebitis* (Phlegmasia alba dolens or "Milk leg"). This is usually seen post-partum or postoperative, causes prolonged incapacitation, may be followed by chronic swelling, induration, and ulcers and *only rarely precedes pulmonary embolism.* In addition to the venous and lymphatic obstruction there may be evidence of sympathetic hyperactivity (coolness and sweating).

During the acute stage the extremity is kept elevated but is not immobilized. We have discarded the ice bag entirely and now use either massive hot moist compresses or a cradle with radiant heat to the lower abdomen and thighs or an electric pad to the abdomen. When the temperature is normal and the edema is minimal, a woven bandage is applied and the patient gradually becomes ambulatory. Subsequently the swelling, depending on its degree, may necessitate an elastic stocking, a latex bandage or a canvas boot.

Two recent innovations in therapy should be mentioned:

a. *Intravenous heparin* has been shown to prevent thrombosis but has the disadvantage of expense and difficulty of administration.

b. *Repeated novocaine block of the lumbar sympathetic ganglia.* It is claimed that the temporary paralysis of the sympathetic outflow produces relief of symptoms and prevents late postphlebitic manifestations. It is a procedure which requires experience. We believe that heat may adequately overcome the vasospasm which is part of the syndrome.

2. *Superficial Thrombophlebitis in Non-Varicose Veins.*

This is a bizarre condition recurring without any known cause. It may follow trauma or exposure to cold weather or it may be related to focal infection. About twenty-five per cent of patients with Buerger's disease have migrating phlebitis.

When there is marked discomfort or fever we advise bed rest with elevation and heat. If the process extends above the knee, division of the saphenous vein at the groin is performed. Otherwise, these patients are encouraged to be ambulatory.

Figure 2

Figure 3

3. *Thrombophlebitis in Varicose Veins.*

This is a common complication of varicose veins. Here again the patient is put to bed only if the thrombophlebitis is disabling or is associated with fever; heat and elevation are then used. Ligation at the saphenofemoral junction prevents embolism and seems to hasten resolution.

4. *Peripheral Deep Thrombosis.*

Thus far we have discussed three types of phlebitis in which there is likely to be an inflammatory reaction in the vein wall, some clinical reaction, an adherent thrombus and thus less chance of embolism.

The following syndrome is more treacherous. Clotting apparently begins in the veins among the calf muscles and may progress, sometimes rapidly, to the upper femoral region, or even higher. It is the loosely attached thrombus or its free-floating propagating tail which breaks off and journeys to the heart and lungs.

We believe that this type of *bland thrombosis* occurs more frequently than has been recognized and that it is the great source of pulmonary embolism.

The onset is usually mild and insidious with little or no change in the chart. Most of the patients have felt pain in the leg, usually in the calf, occasionally in the heel or popliteal region. The pain may be mild or severe. One patient *after an infarction* admitted mild pain of four days duration. In twenty-five per cent of our cases the diagnosis was made after the infarction. On physical examination, there is tenderness in the calf and forced dorsiflexion of the foot causes discomfort in the calf or behind the knee (Homans' Sign).

Because we have witnessed several fatal embolisms during a waiting period of indecision, we now feel justified in dividing the femoral vein, distal to the profunda, whenever the diagnosis of deep calf thrombosis can be made. The patient becomes ambulatory in a few days if there are no general contraindications. Some type of support is then advisable because of the tendency to slight swelling, due probably to the deep thrombosis rather than the ligation.

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BAKELITE BRAINS

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We have prepared as permanent dry specimens 25 bakelite brains. By modifying Rosett's original method[1] we have succeeded in obtaining specimens in which details of brain structure stand out more sharply. Contrast between gray and white matter is distinct; blood and blood vessels are purple black; necrotic tissue is more yellow than normal tissue. Structural details of brain tumors are clear. We have preserved brain dissections as well as flat slices.

Bakelite brains are excellent for teaching. They make the best photographs. They are pleasing to look at and are often mistaken for artificial brain models. Students like to study them, and can handle them for hours without getting eyes, nose and hands irritated by formalin.

The Bakelite Process.

The method consists in infiltration of brain tissue with a solution of synthetic phenol resinoids. Heat polymerizes the resinoids and converts the specimens to firm, dry, hard pieces of bakelite.

Infiltration: Whole brains, dissections, or slices may be used. Slices should be at least ¾ of an inch thick. The specimens are fixed in 4% formaldehyde for 14 days and then transferred to 40% formaldehyde for 7 days. They are then soaked for 30 days in the solution of synthetic resinoids made as follows:

4530 Gm. of Phenol crystals are melted by heating, and to this is added 15 Gm. of sodium hydroxide and 15 Gm. of salicylic acid, previously dissolved in a few cubic centimeters of water. 1684 Gm. of trioxymethylene crystals is added to this liquid. It is important to heed Rosett's caution about the preparation of the resinoid solution. "Its temperature begins to rise after the addition of the trioxymethylene, at first gradually, then with increasing rapidity until, in the course of an hour or two, depending on the temperature of the room, unless precautions are taken, it fulminates, evolving great quantities of formaldehyde gas. If fulmination has proceeded to the stage at which the solution becomes viscous, the chances of stopping up the plumbing should be borne in mind. A practical way of avoiding this is to place the jar in which the ingredients are mixed in a large bucket full of running cool water. The ingredients will crystalize in the course of an hour or two, but the jar should be kept in the running water for twenty-four hours, after which the danger of fulmination is passed. The ingredients will dissolve and combine in the course of a week or two. The process may be expedited without danger after the first twenty-four hours by placing the container in a bucket of warm water. When ready, the solution is clear, transparent, and of the reddish-yellow color of light amber."

Specimens remain in this solution of resinoids 30 days or more. A slice ¾ inch thick is infiltrated in 30 days. A cerebral hemisphere requires at least 2 months. When infiltration is complete, the specimens are translucent, pale brown, and rubbery.

Polymerization: The specimens are taken out of the solution, blotted dry, and placed in a thermostatically controlled oven at an initial temperature of 50°C. To prevent warping stiff brass wires are inserted into the brain substance for support. In the oven the brains and dissected specimens are supported in cloth-cradles slung on wire baskets. By using cloth-cradles distortion of the brain is avoided.

Specimens are baked at 50°C for 7 days, 60°C for 7 days, 70°C for 7 days, and 85°C for 7 days. If the temperature of baking is raised too fast, specimens blister. A convenient electric oven we built for this process is described below. At the end of 28 days of baking the specimens are dry hard pieces of bakelite of cloudy purple color. Details of the brain structure are not distinct.

Developing and Finishing: By boiling the specimens in a solution of 0.1% sodium hydroxide and 2% salicylic acid for 2 hours the purple discoloration is removed, and there is a coating of yellow scum. This scum is readily removed from flat surfaces by polishing on a revolving sandpaper wheel.

[1] Rosett, Joshua: A New Method of Preserving Normal and Pathologic Brain Tissue: Arch. Neurol. & Psychiat. 32:513, 1934.

The authors are indebted to the following pathologists for generously supplying the specimens used in this study: Drs. B. Earle Clark and Robert J. Williams, Rhode Island Hospital; Dr. Constant E. Schradiek, Homeopathic Hospital; Dr. Robert Fienberg, State Hospital for Mental Diseases.

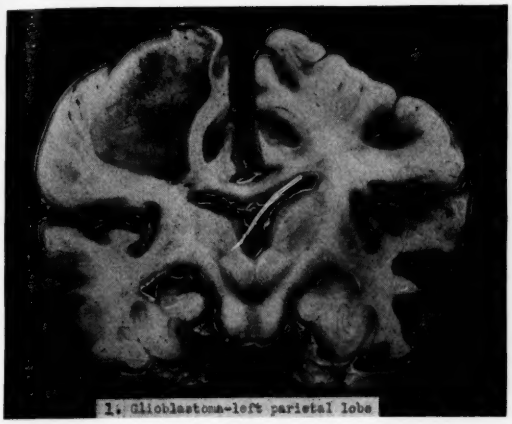

Figure 1

Figure 2

From sulci, crevices, and other irregular surfaces of brains and dissections the scum is removed with a wire brush wheel.

Rosett then polished the specimens by buffing with pumice and lustering with rouge. Rosett's bakelite specimens were striking, but a bit pale and blurred.

We found in our bakelite specimens that better color and greater detail is obtained by putting the specimens into the oven again and baking for 24 hours longer at 85°C. By this additional step contrast in color between white matter and gray matter becomes greater. Tissue differentiation of brain tumors is striking: blood vessels stand out purple black, necrotic tissue is dark yellow, tumor seeding is clear.

Comment: A suitable oven is essential for good results. We have constructed one which is very convenient and inexpensive.

An iron frame 36 inches long, 24 inches wide, and 30 inches high is covered with two layers of galvanized tin and provided with an iron front door hinged to open downwards. Five snugly fitting iron shelves are bolted into the frame 6 inches apart. An oblong slot 12 inches long and 4 inches wide is cut out of each shelf parallel to the end. The shelves are so placed in the oven that the slots are on opposite ends in each succeeding shelf. A small oblong slot 3 by 6 inches is cut in the roof of the oven at the end opposite to the slot in the top shelf. An electric plate is set on the bottom of the oven and wired to a thermostat placed on the top shelf. The thermostat is set at 50°C, thermometers placed on each shelf, the oven door closed, and temperatures read at the end of an hour. The oven is essentially a constant heat-loss apparatus. The bottom shelf is too hot for use, because it receives heat by radiation as well as by convection. If the temperature on the 4th shelf from the top stabilizes at 85°C when the top shelf is 50°C, the 2nd shelf is 60°C, and the 3rd shelf is 70°C. If the temperature of the 4th shelf is more than 85°C, the slot in the roof should be enlarged and the experiment repeated until the 4th shelf stabilizes at 85°C with the top shelf at 50°C. If, on the other hand, the temperature on the 4th shelf is less than 85°C with the top shelf at 50°C, the slot in the roof should be made smaller, until the desired temperature is reached.

Bakelite Brain Specimens.

Twenty-five brains were preserved by the bakelite process. Photographs of three sample specimens are shown:

(1) Figure 1 shows a glioblastoma multiforme in the left fronto-parietal area. Note the distinction between gray and white matter and details of tumor structure.

(2) Figure 2 shows a "butterfly tumor of the corpus callosum". This tumor was a glioblastoma multiforme. It arose in the white matter of one cerebral hemisphere and seeded its way through the corpus callosum to the white matter of the opposite cerebrum.

(3) Figure 3 shows a glioblastoma multiforme in the left temporal lobe. Note the difference in size between the two cerebral hemispheres. The brain and ventricles are markedly distorted.

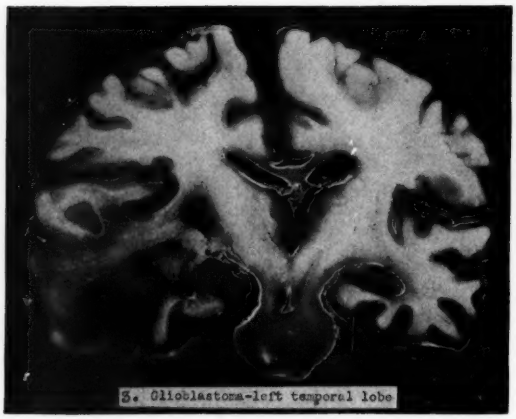

Figure 3

Summary.

By Rosett's bakelite method with Korb modification we have made bakelite preparations of 25 human brains known to us clinically. The process together with the construction of a suitable inexpensive oven are described. Specimen photographs of three glioblastomas are presented.

TULAREMIA

REPORT OF A CASE CONTRACTED FROM AN INFECTED RABBIT TREATED WITH SULFANILAMIDE

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Tularemia was unknown in New England until 1929, and the rarity of its appearance here is not wholly explained. The first case was reported by Dr. Randall,[1] occurring in North Scituate, Rhode Island. A man found three dead rabbits on his farm and contracted tularemia after tearing apart one of the dead rabbits to feed to his cat. The cat died very soon after eating the rabbit, but the patient recovered. In the same year a man in Massachusetts contracted the disease after dressing a cold storage rabbit obtained from the Boston Market.[2] In 1931 in Claremont, New Hampshire, a person became ill with tularemia after dressing two rabbits killed near by. A fourth case occurred in 1933 in the Moosehead Lake region in Maine, where a hunter contracted the disease after skinning a red fox. In 1939 Badger[3] reported the second case diagnosed in Massachusetts. This case was the first reported on the Cape, and was contracted at West Falmouth in 1937. A child ten years old contracted tularemia of the typhoidal type from an infected dog and the mode of transmission of the disease was by the dog's saliva from hand to mouth. Since that time Moss and Evans[4] reported two additional cases of tularemia in Massachusetts, one in 1938 and one in 1939. The latter case was contracted from a wood tick bite. The former was a case registered at the Massachusetts Department of Public Health which was not reported in the literature. Although Rhode Island has not had a reported case since 1929, several dead rabbits were found near the Kimball Bird Sanctuary[5] in South County, in 1937. The dead rabbits showed typical lesions of tularemia and typical organisms were isolated by the Rhode Island State Board of Health. The spleen and liver of an inoculated guinea pig were sent to the National Institute of Health, Washington, D. C., but the organisms were dead on arrival, so that further research could not be carried out to verify the diagnosis made by the Rhode Island State Board of Health. Because rabbits infected with tularemia are prevalent in Rhode Island, and because we should all be on the lookout for tularemia, I felt obliged to report this case.

Case Report.

F. H., a sixty-nine year old widower, white, American, retired pharmacist, was referred to me on November 16, 1940. The complaints were general malaise, headache, slight cough, migratory muscular aches and pains associated with chills and fever of five days duration.

The past history was irrelevant. He has had excellent health and never required the services of a physician.

On November 5, 1940, while gathering bayberry and red elder branches he sustained two puncture wounds on his left hand. Two days later, on November 7, he went rabbit hunting. He shot a rabbit and immediately dressed it. This was the first time he was able to get a rabbit without a dog for several years. The rabbit was thoroughly cooked and eaten by himself, his daughter and son-in-law. He noticed nothing unusual about the rabbit when he dressed it.

Presented as a Case Report at a meeting of the Providence Medical Association, December 2, 1940.

On November 11 he had a severe chill and became ill with a fever of 102.4 degrees. He had general malaise, headache, migratory muscular aches and pains, and went to bed at the insistence of his daughter. Being a druggist he took various simple remedies without benefit.

On November 16, the day I first visited the patient, he was acutely ill with a temperature of 103.4 degrees and a pulse rate of 120, had a slight cough with a severe headache. The muscular aches were much worse. He insisted that he had the grippe and made light of the lesions on his fingers. He was surprised when his physician informed him that his illness was caused by the lesions on his fingers and that he did not have the grippe. He had the ulcers on his fingers covered with small bandages. Physical examination showed a well-developed and well-nourished man who appeared restless and euphoric. His throat appeared red and the tongue was dry. The heart, lungs and abdomen were negative. The blood pressure was 130/86. On the medial aspect at the base of the nail of the left ring finger was an ulcer one centimeter in diameter. It had a reddened base and contained a slough. No pus was present. A smaller ulcer about one millimeter in diameter was found on the outer aspect of the left index finger near the distal phalangeal joint. No red streaks or lymphangitis was present. Several epitrochlear glands of the left elbow were palpated and three large tender glands were present in the left axilla. Motion of the arm caused pain in the axilla.

Laboratory data. The first urine specimen taken showed a least possible trace of albumin, sp. gr. 1018, sugar negative, and sediment showed very few epithelial cells and a rare w.b.c. Three other specimens of urine taken at weekly intervals were negative.

On November 29 the hemoglobin content of blood was 62 per cent, the red cell count was 3,620,000. The red cells varied in size but were consistent in shape. A few cells were very large and extremely achromic. The white cell count was 8,700. The differential count showed 73 per cent polymorphonuclears, 23 per cent lymphocytes, 2 per cent monocytes and 2 per cent eosinophils. On January 10, 1941, the hemoglobin content of blood was 90 per cent, the red cell count was 4,460,000. The white cell count was 8,600. The differential count showed 72 per cent polymorphonuclears, 24 per cent lymphocytes and 4 per cent monocytes. Wassermann and Hinton tests were negative. On November 19, cultures were taken from each ulcer and sent to the Bacteriology Laboratory of the Rhode Island Department of Health where saline suspensions were injected into two guinea pigs.

The first guinea pig died two days after inoculation showing no evidence of infection with B. tularense. The second guinea pig died three days after inoculation. The peritoneal fluid, heart's blood, and spleen showed an abundance of minute gram-negative cocco-bacilli, culturally and morphologically typical of B. tularense.

Blood was taken for agglutination tests and sent to the National Institute of Health on the following days and reported as follows:

November 19—Agglutination of B. tularense: positive in dilution of 1:10, agglutination of Br. abortus: negative.

November 29—Agglutination of B. tularense: positive in dilution of 1:80, agglutination of Br. abortus: negative.

December 4—Agglutination of B. tularense: positive in dilution of 1:320, agglutination of Br. abortus: negative.

December 10—Agglutination of B. tularense: positive in dilution of 1:640, agglutination of Br. abortus: negative.

January 10, 1941—Agglutination of B. tularense: positive in dilution of 1:2560, agglutination of Br. abortus: negative.

On December 4, at a time when the ulcers appeared to be healing, a second culture was taken, and sent to the Rhode Island Department of Health Laboratory where they again isolated the B. tularense organisms and forwarded them to the National Institute of Health. The following is a report from the National Institute of Health: Spleen of guinea pig inoculated from the ulcer of the finger of F. H., and forwarded with your letter of December 6, was inoculated into guinea pigs all of which were dead or dying December 13 with typical lesions of tularemia from which bacterium tularense was recovered.

White blood counts and sulfanilamide determinations were made every other day during administration of the drug. The white blood count ranging between 8,700 on November 29 and 10,100 on December 13, when the sulfanilamide was discontinued. The blood free sulfanilamide determination ranged from 8.0 m.g. per cent to 9.0 m.g.

per cent. The level total ranged from 11.1 m.g. per cent to 11.8 m.g. per cent.

Clinical Course: The patient was very ill and the ulcers and regional glands remained the same. The symptoms and complaints varied little. Pains and insomnia appeared to be outstanding. The temperature fluctuated from 98.8 to 103.8 degrees. Because he showed no improvement from the onset of his illness, he was started on sulfanilamide on November 29, 1940, receiving 90 grains a day with an equal amount of sodium bicarbonate. On December 2, because he had a marked arthritis with painful motion and stiffness of his joints, the dose was reduced to 60 grains a day. By December 10, both ulcers were healed and the temperature remained down running between 98.6 and 99.2 degrees. By December 13 his temperature was normal, and he was free from symptoms except for pain and swelling in his left axilla. At the onset there were three distinct firm palpable nodules in the left axilla. These became confluent and soft. By December 24 there was a flucuent, tender, red swelling the size of a small orange present. This was incised using ethyl chloride spray. About one ounce of pus was liberated. The temperature remained normal. A culture taken at the time of incision was negative for b. tularense. The wound was entirely healed by January 29, 1941.

Definition.

Tularemia is an infectious, but relatively noncontagious disease, due to infection by Bacterium tularense (Pasturella tularense), appearing in man after exposure to the skin, hides or flesh of infected rabbits, rodents, and by the bites of blood sucking ticks or other insects who have previously fed on animals infected with B. Tularense, characterized clinically by a prolonged course, and pathologically by the formation of infective granulomata which occur generally in the reticulo-endothelial tissue.

History.

Tularemia, as we know it, was originally confused with bubonic plague, both clinically and bacteriologically. Wherry,[6] in 1908, published a review of this problem and as a result, until 1909, squirrel plague (now known as tularemia) and human plague were thought to be the same disease. In 1910 McCoy[7] showed that there was a distinct difference between bubonic plague and squirrel plague. In 1911 McCoy and Chapin[8] identified the organism of squirrel plague, cultured it, transmitted it to guinea pigs and named it Bacterium tularense (Pasturella tularense), after Tulare County, California, where the disease was first observed. In 1914, Wherry and Lamb[9] reported the first case of human infection with the organism discovered by McCoy and Chapin. Since 1914 tularemia has become widely distributed throughout the United States, so that, every state and the District of Columbia, except Vermont and Connecticut have reported cases. The disease was reported in Japan in 1925, in Russia in 1928, in Norway in 1929, in Canada in 1930, and in Sweden in 1931. Since 1931 cases have been reported in British Columbia, France, Finland, Czechoslovakia, Maravia, Austria, Turkey and Germany.

The literature contains a large volume of clinical, pathological and bacteriological studies of tularemia, but Francis,[10] who was one of the first students of this disease, describes four clinical types, as follows: 1. Ulcero-glandular: The primary lesion at the site of inoculation is a papule of the skin. This papule later ulcerates and is accompanied by enlargement and often ulceration of the regional lymph nodes. 2. Oculoglandular: The primary lesion is in the eye (transmitted from the hands) and is accompanied by enlargement of the regional lymph nodes. 3. Glandular: No primary lesion is found at the site of inoculation, but there is fever and infected regional nodes are found. Later there is a generalized adenopathy. 4. Typhoidal: Persistent fever is the outstanding feature. There is no primary ulceration and no detectable lymph node enlargement. This type is so named because of its similarity to typhoid fever, with which it is often confused. At other times it passes for fever of unknown origin, septic infection and influenza. The pulmonary lesions in the typhoidal form have led to the diagnosis of tuberculosis or of pneumonia. The cross-agglutination with Brucella abortus and Brucella mellitensis have caused it to be called undulant fever. A clinical diagnosis of typhoidal tularemia is rarely possible. Where persistent, unexplained fever exists, however, specific agglutination tests offer the only clue to diagnosis. The typhoidal type is a generalized infection from the start, with minor cutaneous or regional lymphatic involvement, or more commonly none at all. In the other three types of the disease the lymphatic system appears to bear the brunt of the attack. Multiple lesions in the deep organs and lungs characterize the typhoidal type.

The mortality rate of the typhoidal type of tularemia Foshay[11] found to be approximately 40 per cent, nearly four times that of other clinical types, and the incidence of complicating pneumonia four times the average in the other three types.

Source of Human Infection.

Human beings become infected with Pasturella Tularensis by contact with the raw flesh or blood of infected animals, by the bites of blood sucking insects and flies and by the eating of insufficiently cooked infected meat. Francis[2] in a recent report pointed out that twenty varieties of wild life contract and transmit tularemia. The cottontail rabbit, the snowshoe hare and the jack rabbit account for 90 per cent of all human infections.

Most infections occur through wounds or abrasions in the skin, most frequently of the hands, but Pasturella Tularense can penetrate the unbroken skin. Wood ticks, horse flies, dog ticks, deer flies, house flies, fleas and bedbugs have been shown to cause infection. Tree squirrels and opossum have been responsible for cases of tularemia. One case each has been reported from skinning a sage hen, coyote, deer, red fox, and bull snake. Two cases each came from contact with quail, ground hog and skunk. Two patients have been scratched by cats. Single cases have followed from bites of the cat, skunk, coyote, tree squirrel, opossum, hog, lamb, white rat, and dog. Contamination in the latter group of cases is believed by Francis to be from the animals' mouths.

Bacterium Tularense have been recovered from water rats, meadow mice, muskrats, sheep, ring-necked pheasant, ruffed grouse, fox squirrel, cattle, game birds, beavers, sparrows, and domestic fowl. The literature contains papers discussing the above mentioned animals as sources of infection.

Market men, hunters and meat dressers are most frequently affected, usually with the ulcerative type. Laboratory workers have frequently been infected with a high incidence of the typhoidal type of the disease.

Non-Contagiousness and Immunity.

According to Francis there is no record of transfer of infection from man to man. Doctors, nurses and other attendants on the sick have not contracted the disease. One attack confers a lasting immunity. Blackford,[12] however, reports two cases of ulcerative dermatitis where living organisms were obtained five to twenty-one months after the initial infection. Foshay, discussing Blackford's paper, says that relapses, he believes, are due to residual living bacteria in the tissues and most commonly occur in cases with ulcerating skin lesions and chronically suppurating lymph nodes.

Other Etiologic Features. B. tularense apparently attacks individuals irrespective of their age or sex. It is said that the Negro is not very susceptible. The disease exhibits well marked seasonal incidence; in the East it occurs during the open season for rabbits, while in the West it occurs during the warmer months when ticks, flies and other insects are most prevalent.

Pathology. The pathologic changes of tularemia involve chiefly the recticulo-endothelial system. There is a marked lymphangitis which is nodular in type, an exaggerated lymph adenopathy and involvement of the liver and spleen. The condition is essentially that of an infectious granuloma.

Symptoms.

After a short period of incubation, ranging from one to six days, and averaging about three and one-half days, there is a sharp onset of the disease, usually without prodromal symptoms of note. There occurs a severe chill, followed by the prompt elevation of the temperature and associated with pains and aches throughout the body, notably in the muscles of the back and thighs, and associated with headache. The prostration at this time is quite marked, although not as severe, as a rule, as with other infectious diseases that have an equally abrupt onset.

Diagnosis.

Under ordinary circumstances there is not very much difficulty in recognizing a case of tularemia,[13] the ulcer on the finger, the very large regional lymph glands, and the frequent general enlargement of the lymph structures throughout the body, making it possible to readily diagnose the condition. In rare instances it may be that typhoid fever, malaria, tuberculosis, or Hodgkin's disease may simulate or be mimicked by typhoid or glandular types of tularemia, or the ocular type, gonococcic ophthalmia.

In its bubonic forms the disease is one of the easiest of infections to diagnose correctly from history and physical signs alone. The appearance of regional buboes with diameters of three to four centimeters without associated visible lymph-

angitis by the fourth to sixth day of a prostating febrile disease in patients who give histories of contacts with wild animals, ticks, or biting flies is very seldom duplicated by any other infection.

The typhoidal clinical type often presents great difficulty in diagnosis and it is always necessary to resort to laboratory aids for confirmation. More cases of this type would be diagnosed correctly, and earlier, if the possibility of tularemia were borne in mind and if diagnostic tests were applied at once and repeated every four or five days, especially for patients with unexplained fevers and atypical pneumonias that occur during the rabbit, squirrel, and quail hunting seasons, and during the periods of greatest activity of ticks and blood sucking flies. Early and accurate diagnosis is especially desirable since the typhoidal type has a mortality risk four times that of the other types and because these patients respond especially well to the early serum treatment.

The organism B. tularense can most always be recovered from the ulcer. It is a short, non-motile, nonspore-forming rod, which shows well-marked pleomorphism.

The blood count affords but little assistance in diagnosis. The average blood picture in the counts from the literature was: erythrocytes 4,000,000, leukocytes 11,600, hemoglobin 65 per cent. Differential count: mononuclear cells 23 per cent, polymorphonuclear cells 77 per cent. The extremes of the leukocyte count were 34,000 and 3,500.

The Agglutination Test.

The provisional diagnosis of tularemia may be absolutely confirmed by the agglutination reaction which is obscured only by occasional cross agglutination with B. Melitensis and B. Abortus, which reaction may remain positive for a considerable period after the patient has had an attack of the disease.

Francis states that there is apparently a complete absence of agglutinins for Pasturella Tularensis during the first week of the disease. The appearance of specific agglutinins occurs sometime in the second week, with an abrupt rise in titer in the third week, reaching its maximum in the fourth to seventh week. After the eighth week the titer usually falls, but positive agglutinins have been known to remain as long as twenty years.

Foshay states that the reliability of the microscopic serum agglutination test for verification of diagnosis approximates perfection, although many thousands of cases have been studied in this and in other countries, there is no report of a single failure of a patient to develop serum agglutinins unless death occurred during the first two weeks of the disease. Moreover, once agglutinins have been acquired as a result of infection they have not yet been found to disappear completely thereafter, even if tested for as long as thirty-three years after recovery. The use of this one test, therefore, makes it possible to diagnose the disease, both in its acute phase and after recovery, with a degree of accuracy seldom encountered in other infectious diseases.

Although the serum agglutination test is extremely reliable the well-established knowledge that serum agglutinins never appear during the first week of disease, and occasionally may not appear until the third week or, rarely, until the fourth week, makes it unsuitable as a test for early diagnosis. Nevertheless, the inevitability of eventual appearance of agglutinins makes this test an ideal standard with which the results of other diagnostic aids can be compared.

Skin Tests. Foshay[13] has shown that an intradermal test done with oxidized bacterial suspension of B. Tularense is the earliest diagnostic aid for determining the presence of tularemia. The allergic skin response is specific and reliable. Positive reactions occur as early as the fourth day of illness, almost a week before agglutinins appear in the blood. He has demonstrated that positive skin reactions are constant in the presence of tularemia, but do not occur in normal persons or in the presence of other acute infections.

The test is performed by injecting intracutaneously about 0.05 cc. of the B. Tularense suspension, to make a wheal 5 m.m. in diameter. It should be read forty-eight hours after the injection is made. The special antigen is safer than a highly diluted suspension made from formaldehyde-killed agglutinating antigen, since the latter is capable of provoking occasional serious focal and constitutional reactions as well as necrosing local reactions. Falsely positive reactions have not yet been recorded with Foshay's skin test antigen, and the test is highly specific. A positive reaction has the appearance of a positive tuberculin test and, like the latter, usually requires forty-eight hours for its development. No delayed reactions, appearing after forty-eight hours, have been noted.

The chief advantage claimed for the skin test is that it will usually confirm the diagnosis during the first week of disease when serum agglutinins are always absent.

Cutaneous tests with diluted anti-tularemia serum are performed by injecting intradermally 0.04 cc. of the diluted immune serum at one site and the same amount of diluted normal serum at another site. It is imperative that the control tests be made every time the immune serum is used in order to exclude the possibility of previous serum sensitization. Positive reactions appear almost immediately in most cases, seldom later than the third minute and never later than the sixth minute after injection. This reaction should be read immediately and at five-minute intervals up to the twentieth minute. A positive reaction consists of enlargement of the central wheal to two or two and a half times its original diameter, with a surrounding areola of erythema usually at least one inch (2.5 cm.) in diameter, at the site of the immune serum injection, with no reaction at all at the site of the normal serum injection. Falsely positive reactions have not been recorded, and the specificity of the test appears to equal that of the cutaneous test with the bacterial antigen.

Both of these tests have given positive reactions when applied as early as eight hours after the initial chill. Foshay does not recommend the use of the antiserum test for general diagnostic use until proper test and control serums can be made available.

Prognosis.

The average patient who has tularemia will recover. Throughout the country as a whole the mortality rate is 3.75 per cent. Although the prognosis is excellent as to life, it should not be forgotten that a profound psychic and physical depression persists for many weeks and months after the original infection, and that during this time there is a likelihood of the development of some intercurrent infection which may be very much more severe than in the normal individual and which, in turn, may be responsible for death.

Treatment.

The antiserum developed by Foshay[18] is of specific therapeutic value. There is, with its use, a definite antitoxic action with a fall in temperature, disappearance of symptoms and regression of lymph nodes, if present, and healing of primary lesions.

In Foshay's[13] most recent article he has definitely shown by his figures that serum therapy shortens morbidity and diminishes mortality to significant degrees. Serums were prepared from goats and from horses by prolonged series of subcutaneous inoculations with dense formaldehyde killed suspensions of virulent strains of B. tularense. The usual mode of administration of the serum was by the intravenous route. Many patients received it by intramuscular injection and most children were treated by subcutaneous injections.

Symptomatic treatment of tularemia still holds the place it should in any acute infection, but specific serum now offers the safest and surest method of treatment. Intravenous chemotherapy with arsenicals[14] and iodides[15] has proved of no value. X-ray treatment[16] for primary local lesions has been thought to shorten the course of the disease if applied in the first four days of the disease, but is of doubtful value.

Immunotransfusions has been shown of value, but suitable donors are difficult to find. Foshay[13] reports a case of acute tularemia treated as effectively by immune serum as by antiserum.

No accurate data are available on the use of sulfanilamide in the treatment of tularemia.[15] Unpublished information on its use in thirty cases indicates little or no favorable action of this drug on tularemia.

Curtis[17] reports successful results with sulfanilamide treatment in four cases. He states "four cases of tularemia were successfully treated with sulfanilamide, which was first administered more than a week after the beginning of the disase. In one case sulfanilamide was given within twenty-four hours of the acute onset of the illness and it did not prove beneficial. It is the present consensus that the best results are obtained in the treatment of infections with the gonococcus and the various forms of streptococcus when sulfanilamide is not started until the body has had time to elaborate its own defense mechanisms. Analagously, in my opinion sulfanilamide should not be used during the first few days of tularemia but it is invaluable when instituted after the defense mechanisms of the host have been brought into play."

Treatment therefore consists of prophylaxis and curative measures. With the extensive dissemination of the disease among the rodent population of this country, notably the rabbits, preventive meas-

ures to combat the spread of tularemia have to do largely with the proper handling of rabbits that are killed by hunters, and are used by them in their homes, or if market hunters send the animals to be sold to the general population for food and the skins to be used for wearing apparel, those engaged with the skinning of the animals, the handling of the fur, and the preparation of the animal itself for market should take precautions which will prevent the development of the disease. They should protect themselves with rubber gloves and frequent careful washing of hands. Possible hunters and market men who handle rabbits from grossly infected areas should be immunized. Foshay[13] urges this for those doing laboratory work or animal experimentation, feeling that it is the only prophylactic measure of value. Bacteriologists who handle the organisms frequently develop the disease despite elaborate precautions that have been taken to prevent infection. In the far West, men whose occupations are such that they are exposed to the bites of ticks develop the disease with a fair degree of frequency. They should wear protective clothing. In the case of occasional hunters, they should be warned to avoid shooting or killing rabbits which are sluggish and which do not make the customary effort to get away from the hunter. Such rabbits should be avoided like the plague.

The glands should never be incised, unless there is definite evidence of suppuration, and even then there is considerable dispute as to whether the glands should be opened when the patient has any fever. It is best to let these glands go for some time and see if resolution does not take place, or if it does not, then at a considerably later period, when presumably they have become sterile, to incise them. One of Musser and Cannon's[18] fatal cases had a fluctuating gland in the neck which the surgeons thought should be opened and drained. Apparently as a result of this procedure, there was a rapid dissemination of the organism throughout the body and death occurred in a comparatively short time. Surgery, therefore, should be used only for the incision of acute suppurating glands.

Summary.

A case of tularemia is reported because of its rarity in the State of Rhode Island. Sulfanilamide undoubtedly shortened the length of this patient's illness. An interesting fact, is the behavior of the incised gland, namely, the rapidity of its healing. At the Charles V. Chapin Hospital an analagous condition was noted in the sulfanilamide treatment of their scarlet fever cases. The physicians noted that the cases that were treated with sulfanilamide and later had glands incised healed more rapidly than when they were not treated with sulfanilamide.

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THE NATIONAL PHYSICIANS' COMMITTEE and CHANGES IN MEDICAL CARE

The National Physicians' Committee has recently sent out literature reaffirming its position and appealing for funds. This committee was formed for the purpose of opposing the "regimentation of medical practice under political domination on a nation wide scale, and for care to any except the medical indigent and those receiving other types of relief." Both the President and Mr. Willkie have stated that they are opposed to the socialization of medical practice. The House of Delegates of the American Medical Association has taken the position which the National Physicians' Committee later adopted. The Rhode Island Medical Society has formally taken a similar stand and as far as we know there is no dissenting minority.

There are still elements of danger in the situation. At present there is no general demand for a government system of medical care. There are however a certain number of people who have a fanatical enthusiasm for furnishing medical care immediately to all the population by government action, and either politicians or well intentioned reformers can use these people to create a demand for increased socialization of medicine. For this reason it is to be hoped that the National Physicians Committee will receive support from a large majority of the profession.

Another danger is that, through antagonism to the so called reformers, the medical profession will fail to provide proper leadership in providing medical care for the poor. The indigent and medically indigent need better care, and it is for the best interests of the community and the medical profession to provide this care without unnecessary delay. For this purpose the medical societies and the individual physicians should be active in cooperating with the tax supported and private agencies in working out such plans.

A much more difficult question is that of our attitude toward voluntary health insurance plans such as the White Cross in Boston and the Group Health Cooperative in New York. The immediate effect of a voluntary health insurance plan is very tempting. The regular payment on an insurance basis is of obvious advantage to both the patient and the physician. On the other hand the ultimate effect of such plans is difficult to foresee. This distribution of responsibility for the individual patient among a group may injure the quality of treatment. Also history has shown that those who most need spreading of the cost of medical care do not in general avail themselves of a voluntary plan and that the pressure for compulsary insurance of all the people soon becomes irresistible. Probably we in Rhode Island shall be wise not to take any fixed position now but to watch carefully the developments of these plans so that we can act wisely when such action is necessary.

We believe therefore, that the physicians of Rhode Island should take the following position.

1. We should furnish active support to the National Committee of Physicians in opposing increasing government control in the practice of medicine.

2. We should cooperate with the local and state tax supported agencies in providing medical care for the indigent and medically indigent.

3. We should watch and study the voluntary insurance plans further before taking a stand on this question.

MEDICAL HISTORY IN NEWPORT

The Pilgrim Fathers had little use and no place for dissenters from their ideas. Episcopalian Holmes suffered thirty stripes rather than pay a fine of thirty pounds; John Clarke was put in gaol and paid a fine of twenty pounds; Roger Williams was banished from Plymouth Colony; the less fortunate Quakers were hung in Boston Common. To escape religious persecution, in the spring of

1636, Williams fled into the wilderness, where, joined by other refugees, he attempted a settlement at Rehoboth, east of the Seekonk River. But warned from this site, which encroached on the holdings of Plymouth Colony, the fugitives crossed the river to the west shore. Williams chose the place which he named Providence while a band led by John Clarke journeyed southward to Aquidneck Island where they purchased land from the Indians and founded the settlement at Newport.

The Puritans enjoyed freedom in Plymouth Colony, Baptists flourished at Providence, but in Newport, as Cotton Mather stated, "Rhode Island was occupied by Antimonians, Anabaptists, Quakers, Ranters, and everything else, and if any man has lost his religion, he may find it in this general muster of opinions." When Quakers were hanging from the trees in Boston Common, the Governor of Rhode Island was a Quaker.

Until 1760 no physician settled in Providence but in Newport the founder had a medical education and had signed his name, "John Clarke, Physician, of London." Dr. John Clarke not only practiced medicine in Newport but on Sundays preached in the First Baptist Church. For a hundred years John Clarke was followed by a line of eminent physicians. The names of Hooper, Halliburton, John Brett, Isaac Senter, Walter Channing, and William Hunter are notable in medical history. Previous to 1772, Dr. Jonathan Easton of Newport was practicing inoculation against small-pox. Dr. Benjamin Waterhouse brought the first knowledge of vaccination to this country when he returned from England in 1800 and first proved its value by vaccinating his own children.

Dr. William Hunter came to Newport in 1752 after completing his studies at the University of Edinburgh under the celebrated Dr. Munro, Senior, founder of the medical school there. Dr. Hunter practiced in Newport for twenty-four years. His apothecary shop, his medical library, and his collection of surgical instruments were famous in their time. In 1755-1756, Dr. Hunter gave lectures on anatomy and surgery in the Senate Chamber of the Old Colony House at Newport. The lectures were advertised in the Boston Post in January and February, 1755. On September 13, 1761, in Trinity Church, William Hunter married Deborah, daughter of Godfrey Malbone, a name celebrated in colonial history. Edward Malbone, painter of miniatures, was a nephew of Deborah Hunter.

About the year 1730, Dean Berkeley created a medical furor in Newport by his advocacy of tar-water as a cure for most diseases. "Tar water is of a nature so mild and benign and proportioned to the human constitution, as to warm without heating, to cheer but not inebriate." Reverend George Berkeley, Irishman by birth, educated at Oxford, had sailed from England with the intent of founding a school for the benighted savages in Bermuda. The captain of his ship could not find the Island of Bermuda but succeeded in discovering a land unknown to him but which proved to be in the vicinity of Newport. Here for some years Dean Berkeley preached in Trinity Church. At Middletown he built a fine residence which he named Whitehall and which he presented to Yale College. In 1730 he founded the Newport Philosophical Society, which sponsored the Redwood Library, oldest American library in continuous use. On his return to England in 1732 he was made Bishop of Cloyne. In 1733 he sent from England the organ which is in Trinity Church. While in Newport Dean Berkeley composed his "On the Prospect of Planting Arts and Learning in America." The last stanza reads,

"Westward the course of empire takes its way,
The first four acts already past;
A fifth shall close the drama with the day,
Time's noblest offspring is the last."

PROVIDENCE MEDICAL ASSOCIATION

March Meeting

A regular meeting of the Providence Medical Association was held at the Medical Library on Monday, March 3, 1941. The meeting was called to order by President Danforth at 8:35 P. M.

The records of the previous meeting were read by the Secretary.

The Secretary reported that the Executive Committee at its last meeting had moved that "Any man known to have been in military service for a period of six months, shall automatically be granted a leave of absence and excused from the payment of dues, which, if already paid, shall be remitted, provided such doctor has not already requested a leave of absence from the Association."

The Association passed a resolution, prepared by the Committee on Pre-School Examination, endorsing such examinations. The resolution follows:

> BE IT RESOLVED, That the Providence Medical Association endorses the pre-school examination of the children of Providence, and that it assures the City Health Department, the City School Department and the Parent-Teachers' Association of its continued support and cooperation in the effort to have all children of pre-school age given an adequate physical examination, preferably by the family physician.

In the absence of Dr. William H. Foley, Chairman of the Legislative Committee, the Executive Secretary read the report of that Committee, which included a summary of all legislation introduced in the General Assembly to date and also included reference to legislation to be sponsored by the Medical Societies.

The President announced that the obituary of the late Thomas J. Smith, prepared by Dr. John E. Donley and Charles F. Gormly, had been filed with the Secretary.

The President introduced Dr. Lucius C. Kingman, President of the State Medical Society, who spoke briefly relative to the mid-winter meeting of the Rhode Island State Medical Society to be held on March 20. He also called the attention of the members to the meeting of the State Society in May to be held in Newport this year. The importance of membership in the State Society was stressed by Dr. Kingman, who called attention to the fact that approximately 150 members of the Providence District Society have not joined the State Medical Society. He pointed out the value of a strong, united organization in these troubled times and urged that every doctor accept the responsibility of aiding in the work of his State Medical Society.

There being no reports of cases nor presentation of specimens, Dr. Danforth introduced as the first speaker of the evening Dr. Gordon J. McCurdy, who presented the topic "Endaural Mastoidectomy." This was explained as mastoidectomy through an incision of the ear canal. The operative technique in simple and radical mastoidectomy was clearly outlined with the aid of slides. Patients were demonstrated, illustrating the early and late appearances of the ear after operation. The paper was discussed by Drs. Adams, Bolotow, and O'Connor.

The second paper of the evening, entitled "Industrial Medicine", was presented by Commander Charles R. Oliphant, Medical Corps, Naval Torpedo Station, Newport, R. I. The speaker pointed out the many phases and purposes of this increasingly important specialty and emphasized particularly the preventive and public health aspects. He also analyzed in detail the tremendous savings to employers made possible by keeping employees at work through the intelligent application of industrial medicine. The paper was discussed by Drs. Cianci and Chaffee.

The meeting adjourned at 11:00 P. M. Attendance, 85. Collation was served.

Respectfully submitted,

FRANK B. CUTTS, M.D., *Secretary*

NEWPORT COUNTY MEDICAL SOCIETY

On Wednesday, January 19, 1927, this being the ninetieth birthday of Dr. William W. Keen, who has served Rhode Island with special distinction, the Newport County Medical Society held its Annual Meeting. The meeting took place in the Senate Chamber of the Old Colony House, where in 1736, Dr. William Hunter delivered the first course of lectures on medical science ever given in the American colonies.

The Annual Address was delivered by the President, Dr. William S. Sherman, on the subject "Early Medicine and Surgery in Newport County." Dr. Sherman said in his address that the corner stone of American Medicine and Surgery was laid in the County of Newport and spoke especially of Dr. Samuel Fuller, who came over in the Mayflower, Dr. John Clarke, Dr. Robert Jeffreys, Dr. John Cranston, Dr. Henry Groonland, Dr. Simon Cooper, Dr. John Brett, Dr. Thomas Moffat, Dr. Benjamin Church, Dr. William Hunter, Dr. Benjamin Waterhouse, Dr. James Gardette and Dr. John Oliphant as among the noted doctors and chirurgeons of early days.

Dr. Sherman said, in part:—

I believe in the past, for out of it has proceeded all that we have and are.

With our settlers, in 1638, came Dr. John Clarke, the first physician of our colony, and one of the most remarkable men of his time. Fresh from the clerical and medical studies at the University of Leyden, he became the pastor of the First Baptist Church in Newport, which now bears his name. It

Newport Herald, Thursday, January 20, 1927. R. I. Medical Journal, XIV: 76, May, 1931.

was due to his work, covering twelve years in England, that the Royal Charter of 1663 was secured. It remained our form of government for one hundred and eighty years. It has been said, "Had Dr. John Clarke of Newport no other claim to the first place among the founders of American colonies, the Royal Charter of 1663 would confer this honor."

In the rooms of the Newport Historical Society is a tablet bearing the following inscription:

TO
JOHN CLARKE, PHYSICIAN,
1599 - 1676
FOUNDER OF NEWPORT,
AND OF THE CIVIL POLITY OF RHODE ISLAND.
ERECTED BY THE NEWPORT MEDICAL SOCIETY.
DEC. 1885.

One of the first acts under the Royal Charter is of more than passing interest as it is believed to be the first medical degree conferred in any of the colonies, March 1st, 1664, in Newport, more than a hundred years before King's College in New York first conferred the degree of Doctor of Medicine.

"Whereas the Court have taken notice of the great blessing of God on the good endevers of Captayne John Cranston of Newport, both in phissicke and chirurgery, to the great comfort of such as have had occation to improve his skill and practice, &c. The Court doe therfore unanimously enacte and declare that the said Captayne John Cranston is lycenced and commistioned to administer phissicke, and practice chirurgery throughout this whole Collony, and is by this Court styled and recorded Doctor of phissicke and chirrurgery, by the athority of the Generall Assembly of this Collony."

This century (1730-1830) was one of great progress in medical education in Rhode Island. Bishop Berkeley, who came to this country to promote education and to establish a college, was a strong influence in the Philosophical Society of Newport, founded at this time (1730). His stay on Rhode Island and his writings made a lasting impression on the world of letters. The good dean must have been interested in the ills of the body as well as those of the mind and soul. Here is some of his advice regarding Tar Water.

"To render Tar Water as generally useful as possible, I would draw up some rules and remarks, in a small compass.

"Norwegian tar being the most liquid, mixeth best with water. Put a gallon of cold water to a quart of this tar, stir and work them very strongly together, with a flat stick, for about four minutes. Let the vessel stand covered forty-eight hours, that the tar may subside. Then pour off the clear water, and keep it close covered, or rather bottled, and well stopped, for use.

"I must own myself persuaded, from what I have already seen and tried, that tar water may be drank with great safety and success in the cure or relief of most if not all diseases, in ulcers, eruptions, and all foul cases; scurvies of all kinds, disorders of the lungs, stomach, and bowels; in nervous cases, in all inflammatory distempers; in decays and other maladies: Nor is it of use only in the cure of sickness: it is also useful to preserve health, and guard against infection and old age; as it gives lasting spirits, and invigorates the blood. I am even induced, by the nature and anology of things, and its wonderful success in all kinds of fevers, to think, that tar water may be very useful in the plague, both as a cure and a preventive."

About this time several medical men of great ability and learning came to us, notable among them Drs. John Brett and Thomas Moffatt, whose work in the interest of the Philosophical Society and Redwood Library did so much to lay the foundation of the medical department now bearing the name of Dr. David King.

The founding of The Company of the Redwood Library was the next step in the field of education. The Reverend Ezra Stiles, afterward President of Yale College, spoken of as "the most learned man in America," was its librarian for many years. He was largely responsible for the establishment of Rhode Island College — now Brown University, 1764.

Quoting from a diary of the Redwood family: "When the establishment of a college at R. I. was first contemplated, by certain persons of the Baptist society (a people who have panted after learning, in order to be able to give a reason for the faith that is in them) Mr. Redwood was applied to for aid, when he offered to give a thousand pounds sterling, on condition that it would be erected on Rhode Island. But an association of wealthy individuals in the town of Providence, chiefly of the family of the Browns offered still more, which happily fixed its location there."

Speaking of the Redwood Library, the same writer says, "The medical part of them (referring to the books) was excellent; they were amply sufficient to give the medical student complete information of all that was then known in the English language on Anatomy, Surgery, Chemistry, and Botany, together with the history of drugs, and of their various preparation and uses, with the history of the progress of Physic from the time of Hippocrates. It sowed the seeds of the sciences, and rendered the inhabitants of Newport a better read and inquisitive people than any other town in the British Colonies."

One of the most important events of this period was the course of lectures given by Dr. William Hunter in the Colony House. Of him Dr. Waterhouse says: "About the year 1756, Dr. William Hunter gave at Newport, R. I. the first anatomical and surgical lectures ever delivered in the twelve colonies. They were delivered in the Colony House, two seasons in succession. His collection of instruments was much larger than any professor exhibits at this day. Dr. Hunter was a man of talents, well educated at Edinburgh, and a gentleman of taste in the fine arts."

He further says, alluding to Dr. Hunter and Dr. Halliburton: "We doubt whether Boston, New York, or Philadelphia, ever had, at one and the same time, two practitioners of physic and surgery, better educated and more skillful than these two gentlemen."

Another who left the impress of his skill and conscientious investigation upon many pupils and followers is Dr. Jacques Gardette, a contemporary of Lafayette, who was commissioned as a surgeon in the French navy. When the French fleet and army arrived at Newport in 1780 he was induced to engage in the practice of general surgery and dental surgery in this town, where he was well received and found "considerable and congenial occupation."

After the Revolution there was activity along the lines of Public Health. Dr. Jonathan Easton had already introduced inoculation into Newport in 1772, his being the first three cases in Rhode Island.

Dr. Benjamin Waterhouse, who became an outstanding figure in American medicine, was born and practiced here. Later he became a professor at both Brown University and Harvard. He introduced vaccination into America, first vaccinating his son Daniel Oliver, five years of age, in July, 1800. This was the first person vaccinated in the Western Hemisphere.

At this period the work of the Navy claims our attention, and the report of the Battle of Lake Erie by Commodore Perry in 1813 throws much light upon the surgery of that day.

Quoting from his letter to the Secretary of the Navy, he writes:—"Of Dr. Usher Parsons, surgeon's mate, I cannot say too much. In consequence of the disability of both the other surgeons, Drs. Horsely and Barton, the whole duty of operating, dressing and attending nearly an hundred wounded, and as many sick, devolved entirely on him; and it must be pleasant to you, sir, to reflect that, of the whole number wounded, only three have died."

Dr. Parsons writes his own experience as follows:

On board the U. S. Sloop Lawrence, 1813

"Most of the action was supported by this vessel, as you will suppose when informed that out of one hundred and fifty men (our ship's crew), thirty-one of whom were sick previous to the action, we had rising eighty killed and wounded. This vessel was engaged with the enemy's two ships on one side and a brig astern raking us for two hours, all within musket-shot, during which we rendered the ships unmanageable, and when this vessel could not discharge another gun the Commander repaired on board the Niagara; and within fifteen minutes after he arrived within close action with them, the enemy's three vessels struck.

"Unfortunately for the wounded, the two surgeons had been confined for several days with fever, and could render them but little assistance. It has however operated in my favor, as I have had all the amputations to perform; and it affords me the greatest pleasure to reflect that in no case have I failed of the best success.

"During the action I cut off six legs in the cockpit, which were nearly divided by cannon balls.

"I had some narrow escapes for my life during the action. Five cannon balls passed through the room in which I was attending to the wounded. Two of the men I had dressed and laid aside in the commencement of the action were killed, before it closed, by other shots.

"I have had very poor health all the cruise, am reduced to a skeleton, but am recovering. Never will I cruise again on this lake or any other.

"I am now on my way in the Lawrence for Erie, having all the sick and wounded of the squadron on board, and shall continue in the hospital with them till they in a measure recover, and will then direct my course toward Cape Home."

Commodore Perry again selected Dr. Parsons as surgeon of the Java, on his cruise to Algiers, and he sailed from Newport on January 22, 1816. Two weeks later, in a severe storm, several men were thrown from the masts and yards; some were killed, and others required surgical operations. In assisting to move them, Dr. Parsons was thrown between decks, breaking his right knee-pan.

On the homeward voyage the crew suffered with the small-pox. Dr. Parsons inoculated them with small-pox virus, and all had the disease mildly. Some continued sick when the Java arrived in Newport, March 3, 1817, and the town authorities gave permission to "land a number of persons from on board the said ship, with the small-pox, at Coaster's Harbor island."

Dr. Charles Cotton, of Newport, a pupil of Dr. James Thatcher of Plymouth, of Revolutionary fame, is another outstanding figure of the Navy. He was commissioned as surgeon in the U. S. Navy on April 26, 1813. He was in the battle between the U. S. S. Hornet and H. B. M. S. Peacock, when the latter was captured, and is said to have been severely censured by Commodore Bainbridge for unnecessarily exposing himself in the action. February 10th, 1820, he received a silver medal, by act of Congress, for gallant services.

He was on board the Constitution when she carried Hon. John Jay to France. Afterward he was stationed at Charlestown navy yard, and in 1817 had charge of the naval hospital at Newport, R. I.

Dr. William Turner of Newport interests us as one who served his country in a dual capacity. Upon the breaking out of the second war with Great Britain he was appointed by Oliver H. Perry surgeon of his flotilla at Newport, June 29th, 1812, which position he held until September 29th, 1812, when he was commissioned surgeon's mate, U. S. Army, and ordered to Fort Walcott, Newport Harbor. On the 24th of April, 1816, he was commissioned post surgeon, and surgeon on June 21st, 1821. He was the first surgeon attached to Fort Adams, and supervised its sanitary arrangements during construction. He remained on duty at Forts Walcott and Adams until his death, September 26th, 1837.

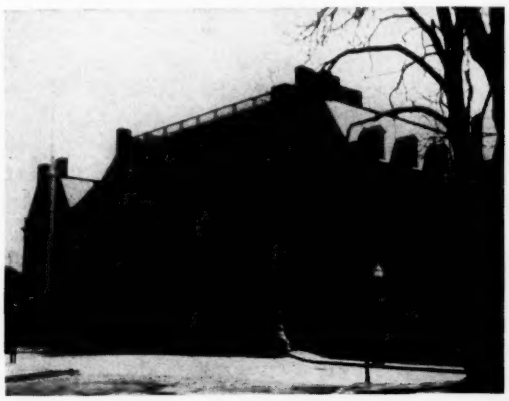
CIVIC CENTER OF NEWPORT

Points of Interest

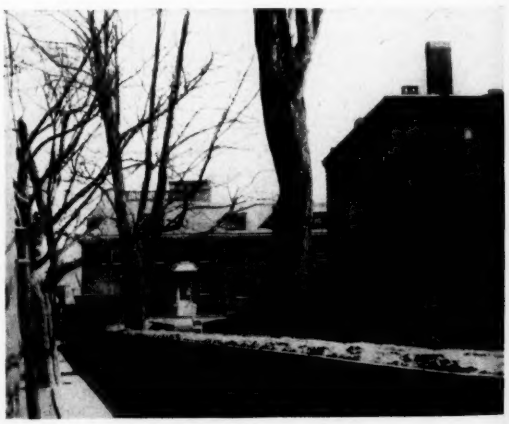
1. The Stone Mill. In Touro Park.
2. Redwood Library. On Bellevue Avenue. Incorporated 1747. Oldest American library in continuous use. Abraham Redwood, Quaker, gave five hundred pounds to start the building fund. The lot on which the building stands was then known as Bowling Green. Dr. Walter Channing often studied here in his youth.
3. City Hall. Broadway and Spring Street.
4. Newport Historical Society. Touro Street. Seventh Day Baptist Church in rear, built in 1729.
5. First Baptist Church. Spring and Stone Street. Founded by Dr. John Clarke.

   Vernon House. Clarke Street. Old murals on exhibition.

   Wanton-Lyman-Hazard House. Broadway and Stone Street. Scene of Stamp Act Riot, 1767.

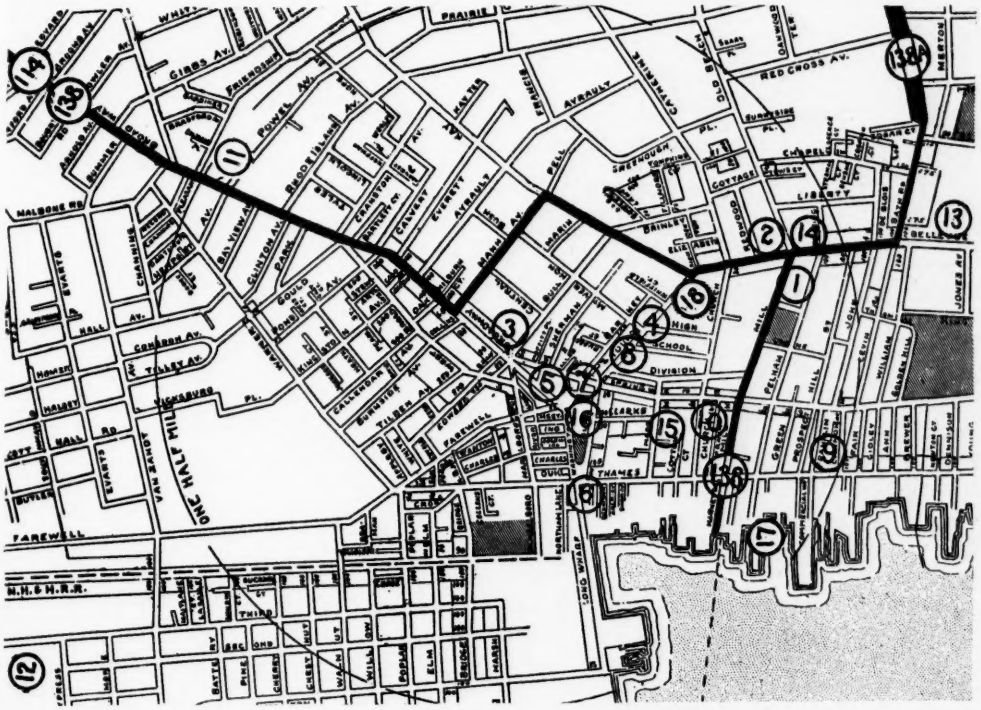


Hotel Viking
Headquarters for 130th Annual Meeting of the Rhode Island Medical Society.



The Newport Hospital. Front on Friendship Street

East



West

6. Old Market House. Thames Street at foot of Washington Square. Occupied by Newport Chamber of Commerce. Call here for Information, Newport City Maps, new booklet "Historic Newport."

   Dr. William Hunter House stood across the street at corner of Thames and Touro Streets. House has been moved to Bridge Street, second house from Washington Street. Dr. Hunter later moved his residence and apothecary shop to the corner of Thames and Mary Streets.

7. Old Colony House 1739-1776, State House 1776-1900. Here Dr. William Hunter gave his lectures on anatomy and surgery in 1755-1756.

8. Synagogue of the Congregation Jeshuat Israel. Touro Street. Oldest synagogue in America.

9. Post Office. Thames and Franklin Street.

10. Trinity Church. Spring and Church Street. Here Dean Berkeley preached. The organ he sent from England. Dr. William Hunter was married and worshipped here. His pew is No. 13. His body lies in the churchyard.

11. Newport Hospital. Broadway, Friendship to Powel Street. Entrance on Friendship Street.

12. U. S. Naval Hospital. From Broadway, turn west on Summer Street. Sign reads "Naval Operating Base, U. S. Naval War College, U. S. Naval Training Station." Follow Van Zandt Avenue across bridge. Turn right on 3rd Street. U. S. S. Constellation moored at Naval Training Station, oldest U. S. warship, built in 1797.

13. Newport Casino.

14. Art Association.

15. Y. M. C. A.

16. County Court House.

17. N. Y. Yacht Club.

18. The Viking Hotel. Headquarters for the 130th. Annual Meeting of the Rhode Island Medical Society.

Route 138A passes by Newport Bathing Beach, beginning of Cliff Walk. St. George's School, Newport Second Beach, Purgatory.

Cliff Walk from Newport Beach to Bailey's Beach, 3½ miles. Several streets lead back from the Walk shortening this distance. At the Newport Beach end, the Walk still shows damage from the 1938 hurricane. The Forty Steps at Narragansett Avenue.

The Ocean Drive. From Washington Square turn south on Thames Street. Right on Wellington Avenue, follow signs along the shore to Bellevue Avenue.

Newport Country Club. Harrison Avenue on Ocean Drive. Apply to Dr. Jacoby for golf privilege.

U. S. NAVAL HOSPITAL, NEWPORT, R. I.

HISTORICAL SKETCH

JOSEPH A. BIELLO, CAPTAIN (MC) U.S.N.

On April 15, 1913, the Naval Hospital, Newport, with a bed capacity of 150, was placed in commission with a staff of four medical officers, one pharmacist, eleven female nurses, twenty-six hospital corpsmen and thirty-two civil employees. Sixty patients from the old wooden building located at the Naval Training Station were received that day for continuation of treatment. The new building was T-shaped, of tapestry brick on hollow tile, and contained two wings of two wards each with the main section allocated to operating suite, sick officers' quarters, messing facilities, and administrative offices.

The location of the hospital is ideal. The original plot of land was acquired in 1909 and consisted of 12.88 acres which was enlarged by purchase of 4.5 acres in 1915 and by transfer from the U. S. Public Health Service of 10.287 acres May 28, 1937. The reservation is located in the northwestern section of Newport about one mile from the business district, and is convenient to bus transportation. The main hospital overlooks the east bank of Narragansett Bay and commands a delightful view of the entire lower bay and harbor entrance. The grounds have a beautiful front lawn stretching to the sea wall, and dotted with small groves of shade trees. Shrubbery groupings are used to break and modify the severe architectural lines, and concrete roads and sidewalks make for easy access to all parts of the grounds.

Once the hospital was put into operation the citizens of Newport realized its value, both commercially and as a decorative asset to the city.

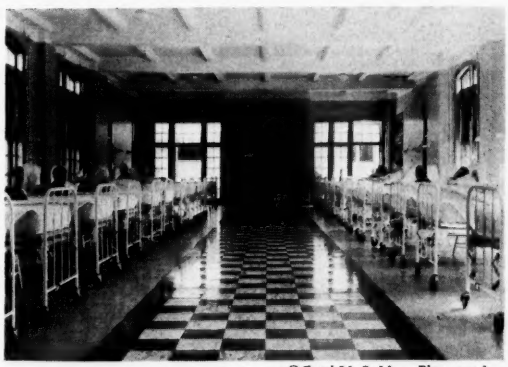
Following America's entry into the World War in 1917 nine wards with a bed capacity of 390 were hastily constructed and the staff had grown to thirteen medical officers, four pharmacists, forty-eight female nurses, and one hundred ninety six hospital corpsmen, while the patient census had grown from 60 in April 1913, to 423 on December 31, 1917. A permanent brick garage was added as were several temporary structures to house maintenance shops, staff personnel, and messing facilities. As Naval activities in this area grew to meet war conditions the hospital kept pace by expansion of its facilities and even during the decimating influenza of 1918 when more than a thousand patients were under treatment at one time, the hospital staff met the emergency and functioned efficiently.

The post-war period was one of gradual retrenchment until the year 1922 when the patient load had dropped to a daily average of 64. With the passage of legislation authorizing admission of Veterans' Bureau beneficiaries to Naval Hospitals the number of patients increased to 240 in December of 1922. This number fluctuated during the years, conditioned by seasons and total apprentice seamen under instructions at the local Naval Training Station. Groups of temporary buildings scattered about the reservation were either razed or so improved as to make them fit into the general architectural scheme of the entire plant.

The need for additional permanent bed space and expanding clinical and laboratory facilities became pressing, and in July 1932 a new northeast wing was added to the main building. This provided fifty-six more beds, a dental department complete with prosthetic laboratory, a modern roentgenologic section and a basal metabolism room. Again, in November 1939, an additional wing was added, which gave 56 more beds, new quarters for a more complete eye, ear, nose and throat department; and an enlarged bacteriological, clinical and pathological laboratory.

The Naval Hospital, in addition to performing its function of keeping men fit for the rigorous requirements of the Naval service, has always answered the call of distress in the community life of Newport. In 1916, when the German submarine U-53 torpedoed British and Canadian merchant vessels off Block Island, this hospital opened a ward for the women and young children survivors brought to Newport by American destroyers and

kept them housed and fed until arrangements were completed for continuation of their journey by rail to New York. In 1917, despite war time conditions, this hospital allocated three temporary wards and furnished staff personnel to care for local civilians, during a diphtheria epidemic, who could not be accommodated at the Newport City Hospital. Again in the late summer of 1925, when the excursion steamer Mackinack suffered a boiler explosion in the upper harbor this hospital threw open its doors, without regard to regulations or red tape, and within half an hour had received about one hundred thirty maimed and injured.



Official U. S. Navy Photograph

U. S. Naval Hospital. A Typical Ward.

Late in 1939, when war again rode the continent of Europe, the Navy once more began to prepare itself against any eventuality. More recruits were sent to the Training Station, necessitating expansion and new construction. This rapid increase in personnel was reflected in a corresponding increase in the patient load of the hospital, and in April 1940 four temporary wards, to accommodate 120 more patients, were erected on the reservation.

At present the hospital has a total bed capacity of 410, with further expansion contemplated. Its staff consists of fifteen medical officers, nine of whom are members of the Navy Medical Reserve Corps; two dental officers, three pharmacists, nineteen female nurses, one hundred thirty three hospital corpsmen, and sixty-two civilian employees. The present Commanding Officer is Captain Joseph A. Biello (MC) U. S. Navy, and the Executive Officer is Captain John T. Borden (MC) U. S. Navy.

Among the Medical Officers of the Reserve Corps on the staff of the hospital are: Commander William A. Stoops, MC-V(G), U. S. N. R., of Newport; Lieutenant Commander Edward L. Merritt, MC-V(S), U. S. N. R., of Fall River; Lieutenant Commander William N. Hughes, MC-V(S), U. S. N. R., of Providence; and Lieutenant Frederick C. Hanson, MC-V(S), U. S. N. R., of Providence.

The complete list of the staff is attached.

MEDICAL OFFICERS

BIELLO, Joseph A., Captain (MC) USN.
BORDEN, John T., Captain (MC) USN.
FORBES, Wilfred W. Lieut. (jg), MC-V(S), U.S.N.R.
HANSON, Frederick C., Lieutenant, MC-V(S), U.S.N.R.
HUGHES, William N., Lieutenant Commander, MC-V (S), U.S.N.R.
HUNTINGTON, Robert W., Jr., Lieutenant, MC-V(S), U.S.N.R.
HUTCHINSON, Rollo W., Commander (MC) USN.
HOEY, Waldo O., Lieutenant, MC-V(S), U.S.N.R.
MacDONALD, Donald H., Lieutenant, MC-V(S), U.S. N.R.
McCANTS, John M., Commander (MC) USN.
O'TOOLE, John B., Jr., Lieutenant, MC-V(G), U.S.N.R.
PERKINS, Paul K., Lieutenant (MC) USN.
STOOPS, William A., Commander, MC-V(G), U.S.N.R.
SUMMERS, Bruce M., Lieutenant Commander (MC) USN.
WEINER, Fred F., Lieutenant, MC-V(S), U.S.N.R.

DENTAL OFFICERS

DELANEY, Edward H., Lieutenant Commander (DC) U.S.N.
PURCELL, James L., Lieutenant Commander (DC) U.S.N.

THE STONE MILL

Historians seem to agree that the Old Stone Mill in Newport was built by Colonial Governor Benedict Arnold to grind corn for the early settlers, basing their opinion on the will of Governor Arnold which refers to "the path from my dwelling house, leading to my stone-built wind-mill."

But certainly this is the strangest wind-mill that was ever built or imagined. A great circular structure of stone supported twelve feet above the ground on eight massive stone columns, it stands on the highest point of land in the vicinity. In the wall to the west, with an unobstructed view of Newport Harbor, was the only window, on either side the wall cut away to give the widest possible view of the bay. Opposite the window is a fireplace with the flue leading up through the thick stone wall.

So, here by the fire would sit the miller, so protected on the land side that nothing could distract his attention from the wide panorama of the sea, viewed from the lofty window, and with no practical way for bringing corn into his mill or for taking the meal away.

Eighteenth Century lithograph of the Stone Mill.

Newport Ruin

W. G. Wall Lithograph of Endecott & Swett, N. Y.

"There are those who think it had been a Fortress but still I can never admit this picturesque Ruin has been a Mill."

From the collection of the Newport Historical Society by courtesy of Mr. Herbert O. Brigham

The Stone Mill is twenty-five feet in height, twenty-three feet outside diameter. It is supported on eight arches resting on stone piers ten feet high. Arches of similar construction crown the large window in the west wall and the fireplace opposite. Evidently the other openings in the walls, lacking these arches, are not a part of the original plan. Just above the supporting arches yawn great square mortises which must have held strong floor timbers. There is no reason to doubt that this structure was built for a watch tower, to sight approaching vessels and to signal them with fire or smoke.

Inside the Stone Mill

Fireplace shows construction similar to that of supporting arches. Openings made later have no arches. Mortise for floor timber at top of each pillar.

Who built the tower we do not know. About the year 1000 the Vikings came from Greenland and made a settlement on this shore. In Icelandic literature the region is often referred to as "Wineland the Good." In 1497 John and Sebastian Cabot explored the coast south to Chesapeake Bay. In 1511 the Portugese Cortereal visited this shore. In 1524 the Italian Verrazzano in his ship "Delfina," explored Narragansett Bay. Of these the most likely to have built the tower at Newport are the Vikings, who are known to have made a settlement and to have visited it several times.

The fanciful impression of Longfellow is given in his "Skeleton In Armor."

"Three weeks we westward bore,
And when the storm was o'er,
Cloud-like we saw the shore
Stretching to leeward;
There for my lady's bower
Built I the lofty tower,
Which to this very hour,
Stands looking seaward."

The Norse Discovery of America. Arthur Middleton Reeves. The Norseman Society, 1906.

RHODE ISLAND MEDICAL SOCIETY

Program for the One Hundred and Thirtieth Annual Meeting

Newport, May 28-29, 1941

WEDNESDAY, MAY 28
MORNING SESSION

Newport Hospital
Friendship Street, Newport

10:30

Auditorium at Nurses' Home

Clinic by members of the Staff of Newport Hospital

12:30

Buffet luncheon will be served to those who have signified their intention of attending the above clinic

WEDNESDAY, MAY 28
AFTERNOON SESSION

Hotel Viking
Bellevue Avenue, Newport

2:00

Call to Order

Welcome by the President

Recognition of Delegates from Maine, New Hampshire, Vermont, Massachusetts and Connecticut

Report of the Trustees of the Fiske Fund

Report of the Committee on Necrology

Papers:

1. Intercapillary glomerular sclerosis or Diabetes-nephrosis syndrome. A report of cases with review of clinical and pathological findings
   B. Earl Clarke, M.D.
   Pathologist, Rhode Island Hospital
2. X-ray Localization of the Placenta and the Clinical Application
   Bertram H. Buxton, M.D., and
   Charles Potter, M.D.
   Providence Lying-In Hospital
3. The Management of the Patient with Chronic Leukemia
   William P. Murphy, M.D.
   Associate in Medicine, Harvard Medical School; Senior Associate in Medicine, Peter Bent Brigham Hospital
4. Biographical Sketches:
   1. Dr. William Hunter
      Roland Hammond, M.D.
   2. Dr. Benjamin Waterhouse
      Herbert G. Partridge, M.D.
   3. Bishop Berkeley's Sojourn in Newport
      John E. Donley, M.D.
   4. Dr. Henry Turner of Newport
      Wilfred Pickles, M.D.

WEDNESDAY, MAY 28
EVENING SESSION

Hotel Viking

8:00

Developments in Medicine and Surgery
Frank H. Lahey, M.D.
Chief of Lahey Clinic, Boston; President-elect, American Medical Association

THURSDAY, MAY 29
MORNING SESSION

United States Naval Hospital
Third and Cypress Streets, Newport

10:30

Naval Hospital

Clinics by members of the Staff of the Naval Hospital

Exhibit of Aviation, Submarine and Industrial Methods and Equipment

This demonstration to be a combined presentation of the Naval Hospital, Naval Torpedo Station, Quonset Point and New London Submarine Base.

12:30

Buffet luncheon will be served to those who have signified their intention of attending the above clinics.

THURSDAY, MAY 29
AFTERNOON SESSION

Hotel Viking

2:00

Papers:

1. Paracolon Bacteria and their possible etiological significance in Gastro-Intestinal Disturbances
   Biographical Sketches
   Professor Charles Stewart
   Biological Department, Brown University
2. The Diagnosis and Treatment of Cancer of the Right Colon
   Leland S. McKittrick, M.D.
   Surgeon, Massachusetts General and Deaconess Hospitals; Chief of Staff, Palmer Memorial; Instructor in Surgery, Harvard Medical School
3. Sex Hormones: Clinical Application
   W. O. Thompson, M.D.
   Associate Clinical Professor of Medicine, Rush Medical College of the University of Chicago; Associate Attending Physician, Presbyterian Hospital, Chicago

Address of the President
Lucius C. Kingman, M.D.

Induction of Officers for the ensuing year

THURSDAY, MAY 29
EVENING SESSION

6:30

THE ANNUAL DINNER will be served at the Hotel Viking

Anniversary Chairman, Samuel Adelson, M.D.

NEWPORT HOTELS

	For 1 Person	*For 2 Persons*		*Suites*
		Double bed	Twin beds	
Hotel Viking, 9 Bellevue Ave.	$3 with lavatory $4 with bath	$5 with lavatory $6 with bath	$7 with bath	Special application
LaForge Cottage, Touro Park West	$3 with bath	None	$5 with bath and up	$8
Muenchinger-King, 38 Bellevue Ave.	$3 with bath	None	$4 with bath and up	$10-$12

NEWPORT COUNTY MEDICAL SOCIETY

President ..... **Samuel Adelson**
First Vice President ..... **Philip Geller**
Second Vice President ..... **Louis E. Burns**
Secretary ..... **Alfred M. Tartaglino**
Treasurer ..... **Norbert U. Zielinski**
Councilor ..... **Charles W. Stewart**
Delegates ..... **Samuel Adelson, Louis E. Burns**

Fellows

Membership in the State Society indicated by surname in capitals

ADELSON, Samuel
Arlen, Richard S.
BURNS, Louis E.
Butler, Maurice
CALLAHAN, James C.
Ciarla, P. P.
Clarke, Philip E.
CORBETT, Francis A.
Creamer, William H.
De Blois, Seth
DOTTERER, Charles S.
GELLER, Philip S.
Healy, John L.
JACOBY, Douglas P. A.
King, Arthur
Lent, J. W.
MacLEOD, Norman M.
McCarthy, Eugene
Ramos, Jose
Redman, William L.
Smith, Daniel A.
STEWART, Charles W.
Stoops, William A.
SULLIVAN, Michael H.
TARTAGLINO, Alfred M.
Tennis, Matthew N.
Walsh, James
YOUNG, John A.
Zielinski, Norbert U.

RHODE ISLAND MEDICAL SOCIETY

President—Lucius C. Kingman, Providence
First Vice President—
Frederic V. Hussey, Providence
Second Vice President—
John Paul Jones, Wakefield
Secretary—Guy W. Wells, Providence
Treasurer—Jesse E. Mowry, Providence
Anniversary Chairman—
Samuel Adelson, Newport
Committee on Annual Meeting—
Walter S. Jones, Providence, Chairman
Charles Bradley, Providence,
Commercial Exhibits
Edward F. Burke, Pawtucket
Ralph L. DeLeone, Providence
Norman M. MacLeod, Newport,
Annual Clinics
Committee of Newport County Medical Society—
Norman M. MacLeod, Chairman
Louis E. Burns, Clinics
Alfred M. Tartaglino, Publicity
Norbert U. Zielinski, Registration
Charles S. Dotterer, Sports

BUNDLES FOR BRITAIN

As much as we individually might be in sympathy with the "Bundles for Britain" movement, one recent phase of it hardly has our approval.

At several points in the country there has been a movement to collect the samples left by pharmaceutical detail men in physicians' offices and include them in the shipments for British Relief. This is an expensive and uncontrolled way of supplying pharmaceutical products.

Most all of the pharmaceutical manufacturers have individually donated supplies with vitamin capsules and other needed pharmaceutical products to the British Relief at no charge.

The packaging of a sample increases the cost and if these samples are collected and sent to Britain, then the purpose for which they were intended, that is, for the use of physicians, is not accomplished, and the heterogenous material that reaches British Relief probably would have little value. Many samples left physicians would be dangerous if used indiscriminately without the advice of a physician.

In some cases individual City and County Medical Societies have been asked to cooperate with the collection of these samples. It is our opinion that such cooperation should be refused for the obvious reasons stated.

National Gastroenterological Association

At the sixth annual convocation of the National Gastroenterological Association, to be held at the Hotel Commodore, New York City, May 13-16, 1941, Dr. Frank A. Cummings, President of the Rhode Island Chapter, will be Co-chairman at the session on Thursday, May 15. Dr. Frederic V. Hussey, Surgeon-in-Chief, Memorial Hospital, Pawtucket, R. I., will speak on the subject "Some Phases of Common Duct Surgery." Discussion will be opened by Dr. Eliot A. Shaw.